Child's Nervous System (2018) 34:1937–1945
https://doi.org/10.1007/s00381-018-3891-x

SPECIAL ANNUAL ISSUE

The spectrum of tuberculosis of the spine in pediatric age group: a review

Sandip Chatterjee[1] · Aditya Banta[1]

Received: 21 June 2018 / Accepted: 27 June 2018 / Published online: 13 July 2018

Abstract

Introduction Pediatric spinal tuberculosis is characterized by rapid bone destruction and carries the risk of rapid onset neurological deficits and severe deformity of the spine. Behavior of spinal deformity over time is affected by growth of spine. Owing to this dynamic behavior of pediatric spinal tuberculosis both in active phase and in healed phase, it presents with challenges which are quite different from adults with caries spine. A clinician must have high index of suspicion for accurate and early diagnosis of spinal tuberculosis in the pediatric population and should also have a thorough knowledge of differences in natural history between adult and pediatric spinal tuberculosis.

Discussion This is based on the senior author's experience of dealing with tuberculosis of the spine in children over the last two decades. Recent advances in field of rapid diagnosis of tuberculosis based on nuclear material-related diagnostic tests have further improved the management of tuberculosis. At the same time, the basic treatment principles remain the same. However, the threshold for surgical vs conservative treatment have subtle differences when compared to adult population. The importance of long-term follow-up after treatment must be appreciated.

Conclusion Tuberculosis in the spine in children needs early attention. Prompting to diagnostic and medical therapy measures can avoid neurological sequellae and delayed deformity.

Keywords Pediatric spinal tuberculosis · Caries spine · Skeletal system

Introduction

The spine is the most common site of tuberculosis in the skeletal system. The destruction of bone and the resultant deformity not only present a problem of altered biomechanics as in rest of skeleton, spinal tuberculosis is further complicated by the presence of neural structures in the vicinity. Pediatric spinal tuberculosis is further complicated by additional factors like more cartilaginous nature of bones, relative rapid destruction, and the effect of growth modulation on the ultimate fate of deformity.

Lack of adequate surveillance for tuberculosis and more specifically for spinal tuberculosis makes an accurate determination of the incidence of spinal tuberculosis an uphill task. The WHO data on tuberculosis prevalence released yearly is the best estimate of disease burden worldwide. However, it depends heavily on the quality of surveillance of member countries. As per the WHO 2017 report, estimated pediatrics population (< 14 years) suffering from tuberculosis in India is 227 (range 99–355) thousand worldwide; this number rises to 1050 (Range 646–1450) thousand [1]. The proportion of the pediatric population having extrapulmonary tuberculosis is typically 20–25% which is higher than the overall incidence of extrapulmonary tuberculosis (16%) [2, 3]. In children, the incidence of spinal tuberculosis is variable. It is reported to be 58% of all spinal tuberculosis in Korea, 33% of patients reported from Chennai (India), and 26% in spinal tuberculosis cases from Hong Kong [4–6].

Anatomical and pathological considerations

The vertebrae develop from 3 primary ossification centers—one for the vertebral body and two for the posterior spinal

✉ Sandip Chatterjee
sandipchat@gmail.com

Aditya Banta
dradityabanta@gmail.com

[1] Neurosciences Division, Park Clinic, 4 Gorky terrace, AJC Bose Road, Kolkata 700017, India

elements. These centers have a central site of bone formation with the surrounding scaffold of cartilage [7]. There are secondary ossification centers for the spinous process, transverse process, facets, and an apophyseal ring at superior and inferior endplate of the vertebral body. Growth not only takes place at the vertebral bodies, but there is growth via expansion of intervertebral disc and annulus too [8]. The growth rate is not uniform during childhood, as the Initial phase of accelerated growth in an infant is followed by a relatively slower rate of growth during 2–10 years of age, and following this is another phase of accelerated growth during the pubertal surge [9].The growing nature of spine and the presence of cartilaginous tissue accounts for some of the major differences in behavior of pediatric and adult spinal tuberculosis.

The infection is caused by *Mycobacterium tuberculosis* and is always a secondary infection, while the primary may be in the lung, genitourinary, or gastrointestinal system. The spread of the bacteria is invariably hematogenous: following a bacillaemia or through the Batson's venous plexus. In 50% of pediatric spinal tuberculosis, the primary site of infection remains unknown [10]. Common primary sites are the lung, lymph nodes, genitourinary, or gastrointestinal system. Mycobacterium is deposited in the anterior aspect of vertebral endplates via the end arterioles. Thus, the anterior type of spinal involvement is the most common form of childhood tuberculosis [11]. The spread of infection may occur to adjacent intervertebral disc, soft tissues, and subligamentous region. Late involvement of intervertebral disc is helpful in differentiating pyogenic and tubercular affection of spine [12]. Soft tissue spread is typically seen in the form of a psoas abscess which may further present as discharging sinuses at atypical locations. Extension of abscess into the epidural region may lead to neurological deficits [11]. Dura, however, is very resistant to spread of infection and this explains the rarity of meningeal symptoms in patients with spinal tuberculosis. The posterior element involvement is very rare. In longstanding disease, multilevel involvement and vertebral collapse resulting in a gibbus deformity are common. This deformity usually presents in active disease. A tubercular lesion which heals in kyphosis may progress over time owing to the growth of spine leading to late-onset paraplegias in healed tuberculosis cases.

Classically four types of spinal bony tuberculosis have been described: the paradiscal lesion, the central type, the anterior type, and the appendiceal type. The paradiscal lesion starts at the vertebral metaphysis and destroys the cartilage and then the disc. The central type is marked by initial infection in the midsection of the vertebral body, and then the infection spreads centrifugally leading to hyperemia and osteoporosis. The anterior lesion has involvement of the body beneath the anterior longitudinal ligament. The appendiceal type is characterized by involvement of the pedicle, lamina, the articular process, or the spinous process.

Presentation

Delay in the diagnosis of pediatric spinal tuberculosis is very common owing to insidious onset and non-specific nature of the initial complaint. All spine tuberculosis cases invariably present with axial pain of varying degree. Pain is associated with muscle spasms, night cries, and sometimes deformity. Sometimes, there may be instability-related symptoms and radicular pain. Cold abscess and discharging sinus may be the presenting symptoms in a subset of cases. Delayed presentation is often associated with a significant neurological deficit.

In children with tuberculosis of the spine, the clinical presentation can be thought of as clinical features of (1) the systemic disease, (2) the bony lesion, (3) the neurological manifestations, and (4) delayed deformity.

Systemic disease

Malaise, anorexia, night sweats, and weight loss are the cardinal systemic features with which children present. These, however, may be completely absent in 60% of children with spinal tuberculosis.

The bony lesion

Localized pain in the spine with limitation of movement occurs due to the bony involvement. There may be severe spasm of the paraspinal muscles, and this is most pronounced in children with atlantoaxial or upper cervical caries who can present with torticollis. Paraspinal abscess, which develops in the early stage of the disease, maybe a retropharyngeal abscess in the neck or in the paraspinal area of the dorsal or lumbar spine, even tracking down to the gluteal region (Fig. 1) or anterior thigh. The pain is often worse at night and is known as "night cries." The reduction of the protective muscle spasm in the night increases the mechanical pain caused by local instability.

Paravertebral cold abscesses occur in over 50% of spinal tuberculosis in children. The presence of abscesses is a sign of active disease. The abscess typically spreads to an area of low

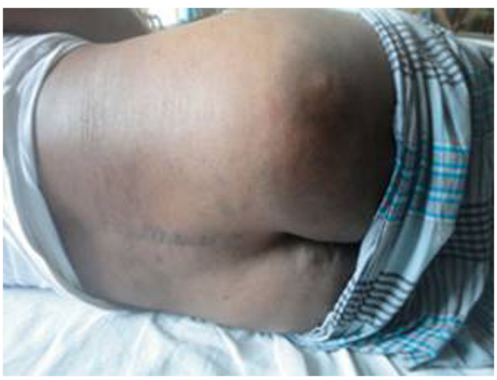

Fig. 1 Cold abscess in the gluteal region

resistance. Hence, in caries of the dorsal spine, the cold abscess may track along the psoas fascia and end up in the groin. Cutaneous fistula as presentation occurs when the cold abscess discharges through the skin (Fig. 2).

Neurological manifestations

The neurological deficit in TB spine is of two types: (1) paraplegia of early onset with active disease usually occurring within the first 2 years and (2) paraplegia of late-onset which occurs years after the healing of the disease or could be because of continued collapse in healed disease focus. Causes of early-onset paraplegia have been postulated to be mechanical compression (abscess, caseous tissues, and granulation tissue) and/or pathological subluxation/dislocation of involved vertebrae [13]. The spinal cord may undergo inflammatory edema and myelomalacia.

Infective thrombosis/endarteritis of the spinal vessels though rare can also be a cause of paraplegia in active disease. Late-onset paraplegia is thought to occur secondary to pressure symptoms from internal gibbus formed after progression of deformity. Additionally, the formation of a constrictive scar after healing of granulation tissue may have a role too [13].

Spinal tuberculosis is associated with slow-growing lesions causing gradual compression of the neural structures. The adaptability of neural structures thus helps maintain neural function after even 2/3rd to 3/4th canal compromise [14, 15]. However, in presence of associated instability and vascular occlusion, neurological deterioration can be seen at lesser canal compromise [16]. Correlation between degree of kyphosis and the severity of paraplegia has not been established in studies though general opinion is a deformity of more than 60 degrees is associated with neurological deficits.

The neurological deficits in tuberculosis spine have been classified by Tuli and modified by Jain into 5 stages [17].

Stage I: patient unaware of the neural deficit, clinician detects plantar extensor, and/or ankle clonus.

Stage II: the patient has spasticity with a motor deficit but is a walker. The anticipated motor score in tetraparesis is between 60 and 100. In paraparesis, it is between 80 and100. The sensory impairment is the lateral column.

Stage III: bedridden spastic patient. Anticipated motor score for quadriplegic is 0–30 and for paraplegic it is 50–80. Sensory scoring is the same as in stage II.

Stage IV: bedridden patient with a severe sensory loss and/or pressure sores. The anticipated motor score in tetraplegia is 0 and in paraplegia, it is 50. There is impairment of both lateral and posterior column sensations.

Stage V: same as stage IV and/or bladder and bowel involvement, and/or flexor spasms/flaccid tetraplegia/paraplegia.

Development of kyphosis

Development of kyphosis in a patient of spinal tuberculosis occurs in active disease due to the destruction of vertebral bodies and resultant collapse of the anterior column [18]. Usually, 3 or more vertebral bodies loose anterior height and lead to an angular kyphosis, nearby vertebral bodies may show inflammatory changes [19]. A truly symmetric destruction would lead to the development of a kyphotic spine (Fig. 3). Asymmetry in this destruction process is responsible for deformity in the coronal plane leading to kyphoscoliotic deformities. The cartilaginous nature of vertebral bodies in children predisposes children to relatively rapid destruction of vertebral bodies. This explains the fact that deformities in children tend to be more severe and also develop in much shorter time as compared to adults.

Another factor that affects the progression of deformity in childhood tuberculosis is the potential for growth in vertebrae after the disease has healed. It must be noted here that effect of growth on spinal deformity is not constant. Growth though previously believed to be a cause of worsening of deformity in majority of post tubercular patient has been found to produce a multitude of effects. Rajshekharan et al. in their study of long-term follow-up of pediatric spinal tuberculosis came up with the findings that 44% kyphosis do not progress, 39% deformity progresses, and 17% remain stationary [20]. Factors such as differential destruction of growth plates by disease process or by surgical debridement, differential loading of spine, and altered vascularity have been implicated in deciding the fate of

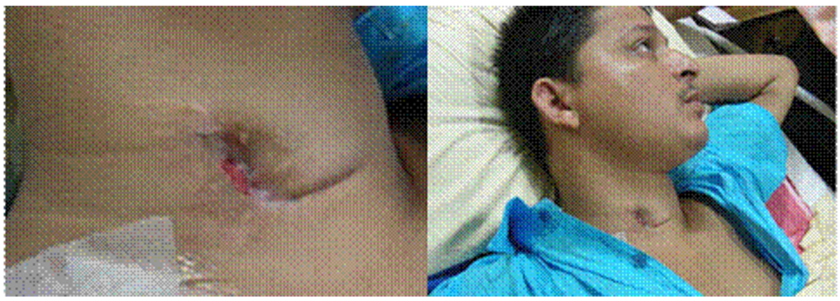

Fig. 2 Tuberculous fistula in the neck following subaxial cervical spine caries

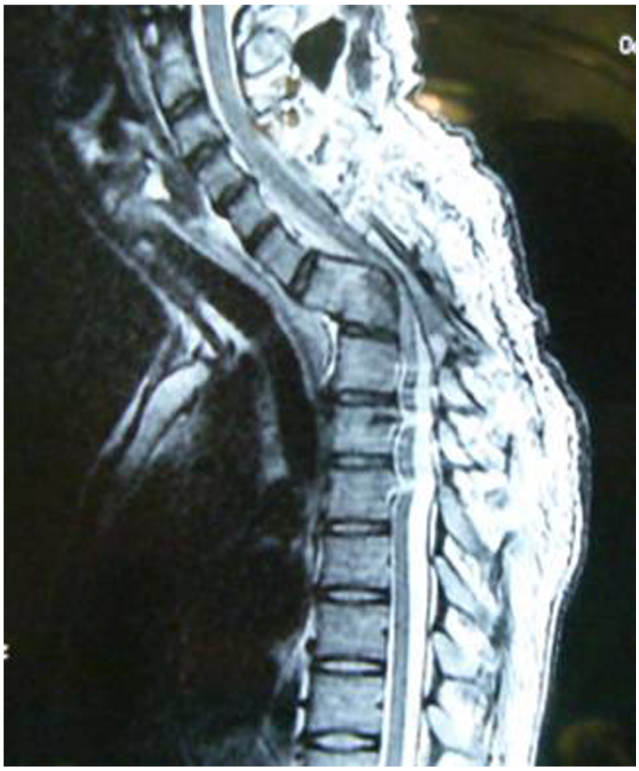

Fig. 3 Kyphotic deformity in cervicodorsal caries spine

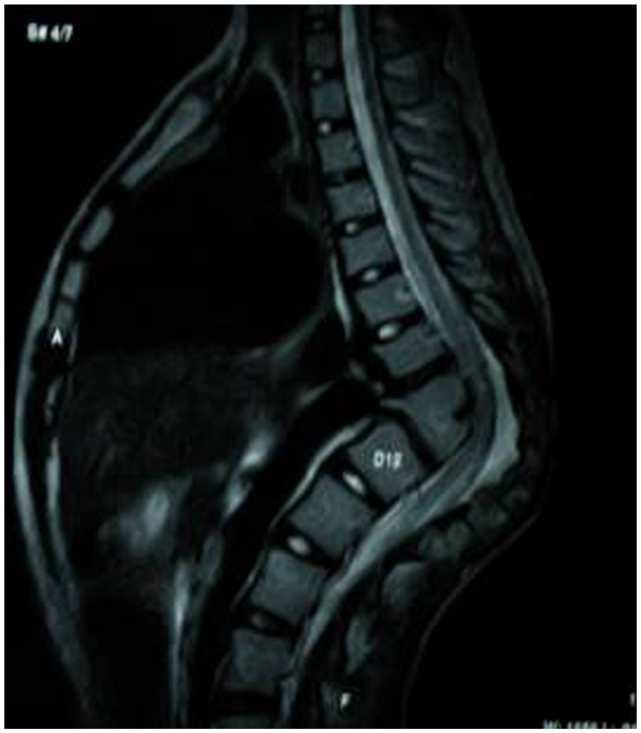

Fig. 4 Stretching of the cord over a kyphotic deformity in late stage of caries spine

kyphotic deformity over time. Rajshekheran et al. also described "spine at risk" signs which can be used to predict the possibility of progression in a particular child with spinal tuberculosis [20]. Progressive kyphosis is associated with increased stretching of cord over the deformity (Fig. 4). Also, as the deformity increases, there is an increase in the size of internal gibbus causing more neural deficits. Additionally, progressive kyphosis and severe spinal deformity in very young children interfere with growth of thoracic cavity and that of the lungs leading to decreased pulmonary functions [19].

Investigating a child with tuberculosis of the spine

CRP is used also to monitor response to treatment. LFT monitoring is recommended to detect adverse reactions to tubercular treatment.

Tests for latent tubercular infection (LTBI) Main value is in regions with low prevalence of tuberculosis. Frequently, these tests for latent tuberculosis are considered diagnostic of tuberculous infection in pediatric population. Patients with LTBI have a 4–6% lifetime risk of developing TB disease, [21, 22]. However, in children, it is 24% and it increases to 43% in infants [23, 24]. Isoniazid reduces the subsequent development of TB disease in patients at high risk [25]. Tuberculin skin test (Mantoux test) detects delayed hypersensitivity to Mycobacterial TB antigens. BCG vaccination results in positive result too. Interferon-Gamma Release Assays (IGRA) have the advantage that there is no cross-reactivity with BCG minimal cross-reactivity with nontuberculous mycobacteria [26].

Tissue diagnosis is the most conclusive way of establishing a diagnosis of spinal tuberculosis in any form. The tissue is obtained either in an open procedure or far more commonly by percutaneous techniques. The conventional culture methods involving use of solid/liquid growth media have a prolonged incubation period and usually give results in 4- to 6-week duration. Additionally, the yield rate of these culture techniques is not high, hence these cannot be relied upon for decision regarding treatment. Nucleic acid amplification test detects mycobacterial genes after gene amplification via polymerase chain reaction, thus giving results in a much shorter duration. A positive result is beneficial when there is clinical suspicion of tuberculosis and a negative result is of little use in excluding the presence of Mtb [26]. Additionally, rapid drug sensitivity testing (DST) can be done for rifampicin and isoniazide resistance. Despite all the new technologies for diagnosis of tuberculosis, diagnosis of spinal tuberculosis must be made on the ground of clinical manifestations and radiology when bacteriology proves negative.

Genotyping of Mycobacterium tuberculosis has epidemiological use in tracing contacts, elucidating sites and patterns of transmission within communities [27, 28]. Clinical use is in determining whether new episodes of TB are due to reinfection or reactivation [29]. These tests are not done routinely [26].

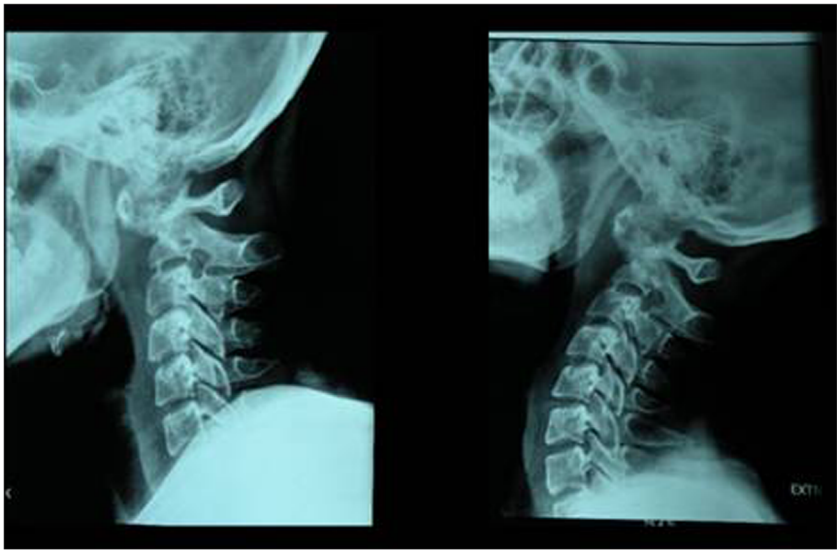

Fig. 5 C1-C2 instability in a case of caries of atlantoaxial joint

Plain radiographs are usually the initial investigation for suspected cases of spinal TB. For a radiolucent lesion to be apparent on a plain radiograph, there must be at least 30% of bone mineral loss. Dynamic Xrays are very important for the documentation of instability (Fig. 5). Computed tomographic (CT) helps in better delineation of bony detail of irregular lytic lesions, sclerosis, disc collapse, and disruption of bone circumference than plain radiograph [30, 31]. CT-guided needle aspiration or biopsy is the technique for early histopathological diagnosis. Radiation exposure is a matter of concern in pediatric population. Magnetic resonance imaging (MRI) is the best diagnostic modality for tuberculosis of the spine and is more sensitive than other modalities. Jain et al. demonstrated correlation between clinical course and MRI observations in patients with tuberculous myelopathy [16]. MRI frequently demonstrates disc collapse/destruction, cold abscess, vertebral wedging/collapse, marrow edema, and spinal deformities. The spinal cord may show edema, myelomalacia, syringomyelia, cord atrophy, thickening of meninges, and/or arachnoiditis with clumping of roots. Compression could be due to extradural fluid collection, caseous material, or granulation tissue (Fig. 6). Patients with preserved cord size with edema/myelitis or fluid collection in extradural space as a cause of compression fare well and can be treated with medications alone. Patients with extradural granulomatous compression on a near normal size cord improve with early surgical decompression. Patients with significant cord compression with evidence of myelomalacia have the least chances of recovery with any sort of treatment [16].

The introduction of newer imaging modalities and nucleic acid-based rapid diagnostic tests has made an early diagnosis of spinal tuberculosis easier, thus decreasing the sequelae of neurological deficit and kyphotic deformity.

Treatment considerations

Tuberculosis of spine is mainly a medical disease requiring surgical intervention in specialized situations only. The

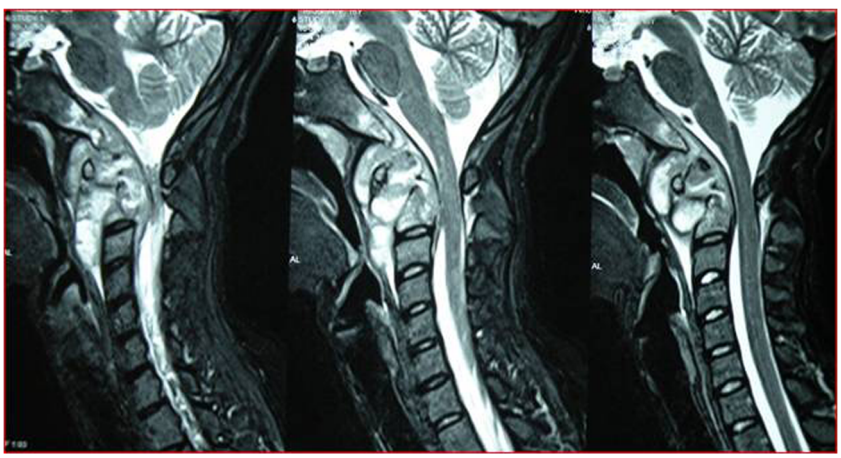

Fig. 6 Extradural compression in caries of upper cervical spine demonstrating prevertebral cold abscess

introduction of effective anti-tuberculous therapy (ATT) has allowed better bacterial control of the disease and healing in TB spine.

Drug regimens used in treatment of tuberculosis in adults and children inherently are the same. However, drug dosage needs to be adjusted according to the weight of the child. Four weight groups are used to decide drug dosage: < 10, 10–17, 18–25, and > 25 kg. Isoniazide and rifampicin 75 mg each, pyrazinamide 250 mg along with ethambutol 200 mg/day are prescribed for a child weighing < 10 kg. Thereafter, these doses are doubled, tripled, and quadrupled for subsequent weight bands respectively [9].

The major concern remains underdosing of individual drugs. As during the course of therapy child tends to gain weight, weight must be measured at regular interval and dosage of drug given as per appropriate weight band. The duration, of course, varies between 6, 9, 12, and 18 months. Studies have shown that 6 to 9 months course of AKT with surgical excision has results comparable to that of 18 months of drug therapy with or without surgery [32–34].

With the emergence of multidrug-resistant tuberculosis especially more in pediatric population, one must actively strive for tissue diagnosis as well as drug sensitivity testing in all patients so that early shift to second line and third line drugs can be made whenever needed. Bracing is an important component of treatment of pediatric spinal tuberculosis as it helps in enforcing movement restriction in otherwise noncomprehending patients.

Surgical treatment is typically reserved for rapid onset dense neurological deficit and neurological deficit which is progressive or which is not improving with conservative treatment, for significant deformity at presentation, and in case of panvertebral involvement (disease of both anterior and posterior column). Significant mechanical instability/potential for future deformity is the other consideration for surgery. Presence of large paraspinal abscesses causing mass effect, persistent pain not improving with chemotherapy, and need for definitive diagnosis are also indications for surgical intervention [35].

Spinal tuberculosis with neurodeficit

Diagnosis of spinal tuberculosis before neurodeficit occurs and subsequent prevention of neurodeficits is the best way forward. Tuli et al. observed neurological parameters in spinal tuberculosis patient on antitubercular treatment and rest therapy while waiting for surgery and found that 30 to 40% of patients showed improvement in neurology during these 4 to 6 weeks [36]. However, this might not be the best treatment for all patients with neurodeficits. In addition to the general surgical indication, early surgical decompression should be considered when MRI shows granulomatous or caseous extradural compression with edema/myelitis of the cord. Presence of myelomalacia is linked to a poorer prognosis but surgery should still be offered. Surgical treatment aims at disease control along with maintenance of stability, normal spinal growth, sagittal alignment of the spine, and ensuring protection of neurological function including providing an environment conducive for neurological recovery.

Owing to the extensive bone loss associated with radical debridement, recent studies have favored a limited debridement and cord decompression [37]. Debridement should be limited to excision of pus, caseous tissue, and sequestra. Inflamed/ischemic and infracted bone tends to recover on starting medical therapy as blood supply to the area improves. However limited decompression should not be done at the cost of inadequate relief of pressure on the cervical cord [16].

Access to cervicodorsal junction area may prove problematic. A transmanubrial approach has been described by the authors [38] to deal with tubercular pathology in this region; a U-shaped manubriotomy is used, thus preserving the sternoclavicular articulation (Fig. 7). This helps in avoiding problems associated with a median sternotomy or with disrupting the sternoclavicular articulation.

Active disease with kyphosis

Surgical treatment is typically reserved for rapid onset dense neurological deficit and neurological deficit which is progressive or which is not improving with conservative treatment, for significant deformity at presentation, and in case of panvertebral involvement (disease of both anterior and posterior column).

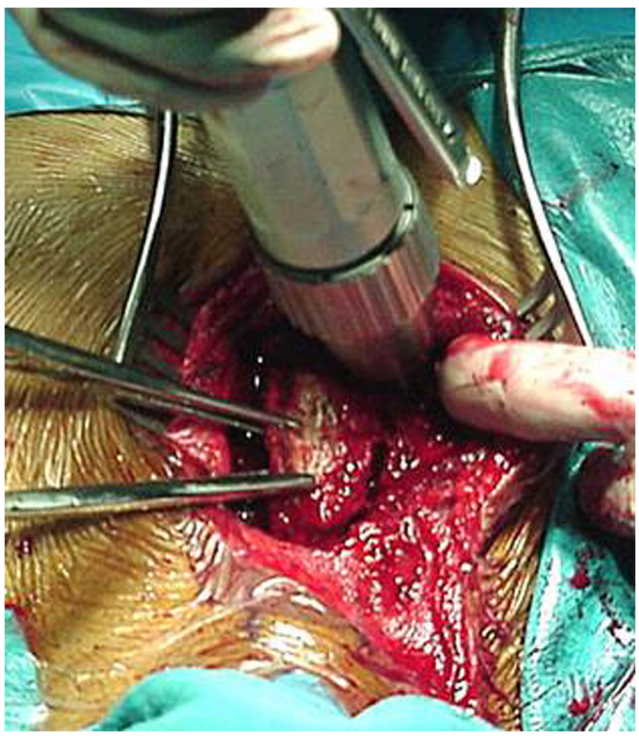

Fig. 7 Transmanubrial approach to cervicodorsal caries

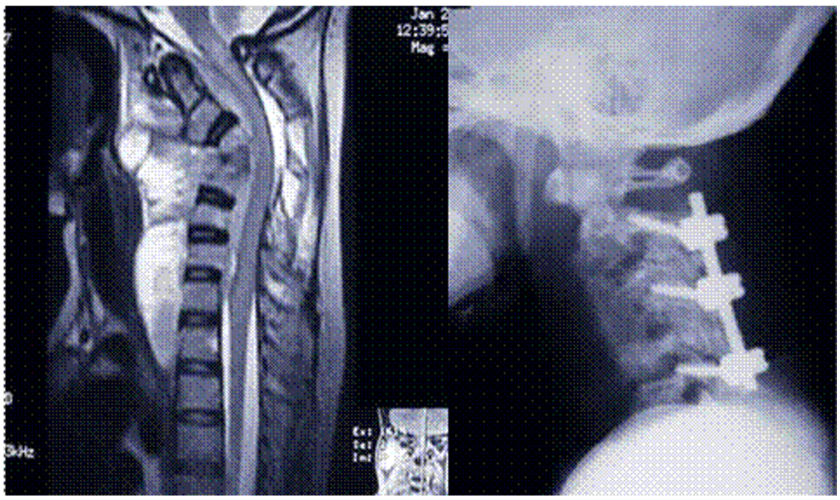

Fig. 8 Anterior corpectomy and posterior fixation in a cervical caries

Significant mechanical instability/potential for future deformity is the other consideration for surgery. Presence of large paraspinal abscesses causing mass effect, persistent pain not improving with chemotherapy, and need for definitive diagnosis are also indications for surgical intervention [35].

All kyphotic tubercular spines with moderate to severe kyphosis (> 50°) may be treated with kyphosis correction surgery because of the inherent potential for neurological deficits in acute and healed stages. The kyphotic spine is associated with shortening of anterior column of spine. During correction of deformity, any acute lengthening stretches the neural structures and hence must be avoided. At the same time, excessive shortening would lead to buckling of cord and neurodeficit. Thus, a balance between the two must be obtained; mild shortening of cord is an acceptable option. The compression is caused by retropulsion of material into the spinal canal, and hence any attempt at deformity correction should be accompanied by neural decompression from the anterior compressing elements as failing to do so would lead to increased risk of neural deficits. The surgery typically consists of anterior corpectomy in addition to posterior decompression (Fig. 8). The procedure as a whole makes the spine highly unstable and both anterior column reconstruction and posterior instrumentation are warranted. Anterior instrumentation are not reliable because of variable hold of screws. Spinal cord should be visualized throughout the procedure [39].

We recommend routine use of intra-operative neuromonitoring for correction of delayed deformity due to cervical spinal tuberculosis. The use of somatosensory evoked potential (SSEP) in addition to monitoring of Trans Cranial MEP (TcMEP) is invaluable in deformity surgery. It acts as a guide to help dictate the extent of the correction.

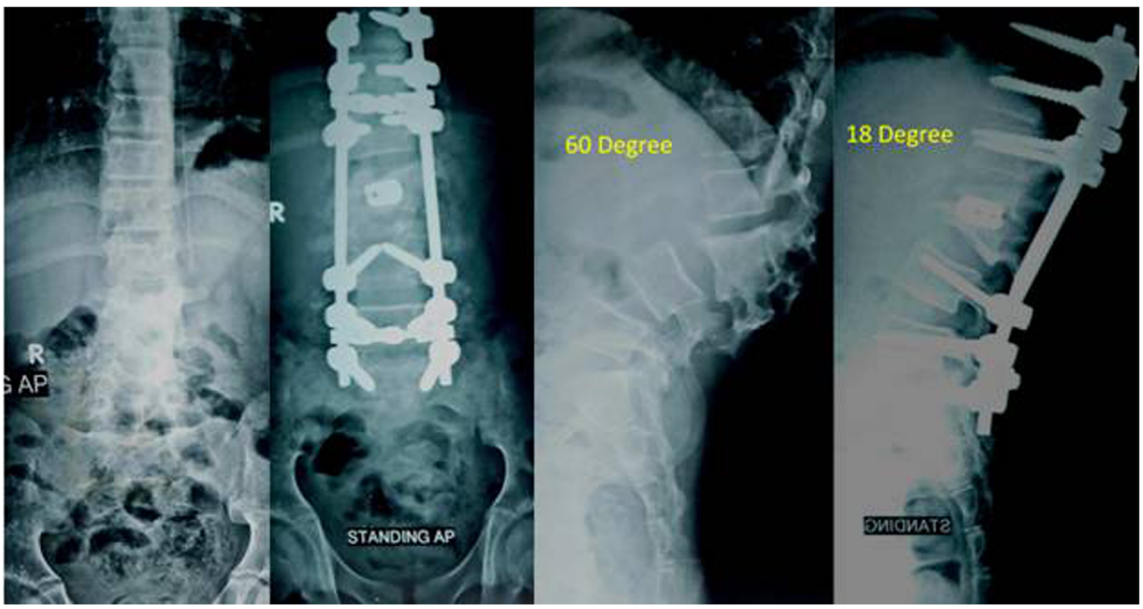

Fig. 9 Pedicle substraction osteotomy in a tuberculous kyphotic deformity

The various methods of kyphosis correction secondary to spinal tuberculosis are:

a. Transpedicular approach—this can be achieved by using a transpedicular decancellation osteotomy or a costotransversectomy and pedicular excision at the apex of kyphosis [40, 41] (Fig. 9).
b. Anterior decompression followed by posterior instrumentation or vice versa. Anterior decompression is done using transthoracic/retroperitoneal approach, the anterior column may be reconstructed by a bone graft or a mesh cage, and this is augmented by a posterior instrumentation which can be performed in the same sitting or at a later stage [42].
c. Extrapleural anterolateral approach—it provides exposure to both anterior and posterior elements at the same time. Using this approach, the patient is operated in lateral position; hence, stability is maintained during the procedure and there is no need for temporary stabilization. Exposure of chest cavity and retroperitoneal structure is not needed.

Kyphosis correction in healed lesion

Correction of kyphosis in healed lesion is a technically demanding surgery and has been associated with higher risk of neural injury; hence, it is not recommended for cosmetic reasons. The aim of surgery in patients with neurodeficit should be decompression and acceptable sagittal and coronal balance rather than attaining normal spinal parameters. When associated with paraplegia of late-onset, anterior decompression and fusion are recommended. Here internal gibbus needs to be removed by approaches similar to those used in active disease. However, when done for healed disease, these procedures are technically more challenging and recovery is less predictable [43].

Follow-up period

Regular monitoring of blood counts, ESR CRP, and liver function is warranted in all patients. The response of these parameters is the major guiding principle for treatment in early course. Radiological investigations in form of MRI should be repeated earliest at 6 months as repeating imaging before caries the risk of misinterpretation of drug lysis as non-responsiveness of disease.

The total duration of antitubercular treatment is highly variable. Our practice is to have a repeat MRI at 12-month duration and stop AKT when the disease shows near complete healing. Persistence of small abscesses is not considered an indication for the continuation of therapy.

Compliance with ethical standards

Conflict of interest Neither author has any conflict of interests.

References

1. World Health Organization. Global tuberculosis report 2016
2. Maltezou HC, Spyridis P, Kafetzis DA (2000) Extra-pulmonary tuberculosis in children. Arch Dis Child 83(4):342–346
3. Moon MS, Kim SS, Lee BJ, Moon JL (2012) Spinal tuberculosis in children: retrospective analysis of 124 patients. Indian J Orthop 46: 150–158
4. (1973) A controlled trial of ambulant out-patient treatment and in-patient rest in bed in the management of tuberculosis of the spine in young Korean patients on standard chemotherapy a study in Masan. Korea. First report of the Medical Research Council Working Party on Tuberculosis of the Spine. J Bone Joint Surg Br 55:678–697
5. (1974) A controlled trial of anterior spinal fusion and debridement in the surgical management of tuberculosis of the spine in patients on standard chemotherapy: a study in Hong Kong. Br J Surg 61: 853–866
6. (1982) A 10-year assessment of a controlled trial comparing debridement and anterior spinal fusion in the management of tuberculosis of the spine in patients on standard chemotherapy in Hong Kong. Eighth Report of the Medical Research Council Working Party on Tuberculosis of the Spine. J Bone Joint Surg Br 64:393–398
7. Basu S (2012) Spinal injuries in children. Front Neurol 3:96
8. Jain AK, Sreenivasan R, Mukunth R, Dhammi IK (2014) Tubercular spondylitis in children. Indian J Orthop 48(2):136–144
9. Dimeglio A, Canavese F (2012) The growing spine: how spinal deformities influence normal spine and thoracic cage growth. Eur Spine J 21(1):64–70
10. Teo HE, Peh WC (2004) Skeletal tuberculosis in children. Pediatr Radiol 34(11):853–860
11. Tyagi R (2016) Spinal infections in children: a review. J Orthop 13(4):254–258
12. De Vuyst D, Vanhoenacker F, Gielen J et al (2003) Imaging features of musculoskeletal tuberculosis. Eur Radiol 13:1809–1819
13. Tuli SM (2016) Tuberculosis of the skeletal system. JP Medical Ltd
14. Hoffman EB, Crosier JH, Cremin BJ (1993) Imaging in children with spinal tuberculosis—a comparison of radiography, computed tomography and magnetic resonance imaging. JBJS 75B:233–239
15. Jain AK, Agarwal AN, Mehrotra G (1999) Correlation of canal encroachment with neurological deficit in tuberculosis of spine. Int Orthop 23(2):85–86
16. Jain AK, Kumar J (2013) Tuberculosis of spine: neurological deficit. Eur Spine J 22(4):624–633
17. Jain AK, Sinha S (2005) Evaluation of paraplegia grading systems in tuberculosis of the spine. Spinal Cord 43(6):375–380
18. Govender S, Ramnarain A, Danaviah S (2007) Cervical spine tuberculosis in children. Clin Orthop Relat Res 460:78–85
19. Jain AK, Dhammi IK, Jain S, Mishra P (2010 Apr) Kyphosis in spinal tuberculosis–prevention and correction. Indian J Orthop 44(2):127–136
20. Rajasekaran S (2001) The natural history of post-tubercular kyphosis in children: radiological signs which predict late increase in deformity. Bone Joint J 83(7):954–962
21. Cohn DL, O'Brien RJ, Geiter LJ, Rockville MD, Gordin FM, Hershfield E, Horsburgh CR Jr, Jereb JA, Jordan TJ, Kaplan JE, Nolan CM (2000) Targeted tuberculin testing and treatment of latent tuberculosis infection. Am J Respir Crit Care Med 161(4):221–247

22. Horsburgh CR Jr (2004) Priorities for the treatment of latent tuberculosis infection in the United States. N Engl J Med 350(20):2060–2067
23. Hoskyns W (2003) Paediatric tuberculosis. Postgrad Med J 79(931):272–278
24. Walls T, Shingadia D (2004) Global epidemiology of paediatric tuberculosis. J Infect 48(1):13–22
25. International Union Against Tuberculosis Committee on Prophylaxis (1982) Efficacy of various durations of isoniazid preventive therapy for tuberculosis: five years of follow-up in the IUAT trial. Bull World Health Organ 60(4):555
26. Lewinsohn DM, Leonard MK, LoBue PA, Cohn DL, Daley CL, Desmond E, Keane J, Lewinsohn DA, Loeffler AM, Mazurek GH, O'Brien RJ (2017) Official American Thoracic Society/Infectious Diseases Society of America/Centers for Disease Control and Prevention clinical practice guidelines: diagnosis of tuberculosis in adults and children. Clin Infect Dis 64(2):e1–e33 5
27. Malakmadze N, González IM, Oemig T, Isiadinso I, Rembert D, McCauley MM, Wand P, Diem L, Cowan L, Palumbo GJ, Fraser M (2005) Unsuspected recent transmission of tuberculosis among high-risk groups: implications of universal tuberculosis genotyping in its detection. Clin Infect Dis 40(3):366–373
28. Kimerling ME, Benjamin WH, Lok KH, Curtis G, Dunlap NE (1998) Restriction fragment length polymorphism screening of Mycobacterium tuberculosis isolates: population surveillance for targeting disease transmission in a community. Int J Tuberc Lung Dis 2(8):655–662
29. Behr MA, Small PM (1997) Molecular fingerprinting of Mycobacterium tuberculosis: how can it help the clinician? Clin Infect Dis 25(4):806–810
30. Moore SL, Rafi M (2001) Imaging of musculoskeletal and spinal tuberculosis. Radiol Clin N Am 39:329–342
31. Gard RK, Somvanshi DS (2011) Spinal tuberculosis: a review. J Spinal Cord Med 34:440–454
32. Guo LX, Ma YZ, Chen X, Bao D, Luo XB (2010) Clinical study of short course chemotherapy in spinal tuberculosis. Zhogguo Gu Shang 23:491–494
33. Kotil K, Alan MS (2007) Medical management of Pott disease in thoracic and lumbar spine: a prospective clinical study. J Neurosurg Spine 6:222–228
34. Parthasarthy R, Sriram K, Santha T (1999) Short course chemotherapy for tuberculosis of spine—a comparison between treatment and radical surgery. J Bone Joint Surg Br 81:464–471
35. Mak KC, Cheung KM (2013) Surgical treatment of acute TB spondylitis: indications and outcomes. Eur Spine J 22(4):603–611
36. Tuli SM (1975) Results of treatment of spinal tuberculosis by "middle-path" regime. J Boone Joint Surg (Br) 57B:13–23
37. Kumar R, Srivastava AK, Tiwari RK (2011) Surgical management of Pott's disease of the spine in pediatric patients: a single surgeon's experience of 8 years in a tertiary care center. J PediatrNeurosci 6(Suppl 1):S101–S108
38. Chatterjee S, Basu S (2003) Trans sternal decompression of postlaminectomy caries of the upper dorsal spine. Br J Neurosurg 17(3):270–271
39. Jain AK, Maheshwari AV, Jena S (2007) Kyphus correction in spinal tuberculosis. Clin Orthop Relat Res 460:117–123
40. Laheri VJ, Badhe NP, Dewnany GT (2001) Single stage decompression, anterior interbody fusion and posterior instrumentation for tuberculous kyphosis of the dorso-lumbar spine. Spinal Cord 39: 429–436
41. Bezer M, Kucukdurmaz F, Guven O (2007) Transpedicular decancellation osteotomy in the treatment of posttuberculous kyphosis. J Spinal Disord Tech 20:209–215
42. Moon MS, Woo YK, Lee KS, Ha KY, Kim SS, Sun DH (1995) Posterior instrumentation and anterior interbody fusion for tuberculous kyphosis of dorsal and lumbar spines. Spine 20:1910–1916
43. Moon MS (1999) Tuberculosis of the spine-controversies and a new challenge. Spine 22:1791–1797